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Exhibit A

- 1. (Amended) A method for improving production of a secondary metabolite by a fungus by increasing the yield of the secondary metabolite in the fungus, the method comprising modulating the expression of a gene involved in regulation of secondary metabolite production in a manner that improves the yield of the secondary metabolite, provided however, that when the secondary metabolite is isopenicillin N, then the modulation is not mediated by transcription factor CPCR1; when the secondary metabolite is sterigmatocystin, then the modulation is not through AfIR, FadA, or FluG; when the secondary metabolite is aflatoxin, then the modulation is not through AfIR; when the secondary metabolite is penicillin and the fungus is Aspergillus nidulans, then the modulation is not through mutations that result in expression of truncated forms of PacC or constitutively active forms of FadA; when the secondary metabolite is lovastatin and the fungus is Aspergillus terreus, then the modulation is not through expression of lovE; and when the gene involved in regulation of secondary metabolite production is from Saccharomyces cerevisiae, then the modulation is not through decreased activity or expression of Hog1, Bem2, Rim15, Sfl1, Ira1, Ssd1, Srb11, Swi4, Tpk3 or though increased activity or expression of Afl1, Dhh1, Inv7, Inv8, Ste21, Pet9, Mep2, Inv1, Inv5, Inv6, Inv9, Inv10, Inv11, Inv12, Inv13, Inv14, Inv15, Cdc25, Mcm1, Mga1, Phd2, Pho23, Ptc1, Rim1, Stp22, Tpk2 or Ypr1.
- 15. (Amended) A method for improving production of a secondary metabolite by a fungus by increasing productivity of the secondary metabolite in the fungus, the method comprising modulating the expression of a gene involved in regulation of secondary metabolite production in a manner that improves the productivity of the secondary metabolite, provided however, that when the secondary metabolite is isopenicillin N, then the modulation is not mediated

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Exhibit A (continued)

by transcription factor CPCR1; when the secondary metabolite is sterigmatocystin, then the modulation is not through AfIR, FadA, or FluG; when the secondary metabolite is aflatoxin, then the modulation is not through AfIR; when the secondary metabolite is penicillin and the fungus is *Aspergillus nidulans*, then the modulation is not through mutations that result in expression of truncated forms of PacC or constitutively active forms of FadA, when the secondary metabolite is lovastatin and the fungus is *Aspergillus terreus*, then the modulation is not through expression of lovE; and when the gene involved in regulation of secondary metabolite production is from *Saccharomyces cerevisiae*, then the modulation is not through decreased activity or expression of Hog1. Bem2, Rim15, Sf11, Ira1, Ssd1, Srb11, Swi4, Tpk3 or though increased activity or expression of Af11, Dhh1, Inv7, Inv8, Ste21, Pet9, Mep2, Inv1, Inv5, Inv6, Inv9, Inv10, Inv11, Inv12, Inv13, Inv14, Inv15, Cdc25, Mcm1, Mga1, Phd2, Pho23, Ptc1, Rim1, Stp22, Tpk2 or Ypr1.